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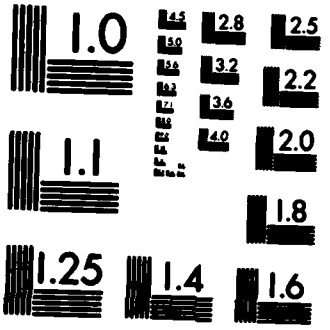

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REPORT NUMBER 3

SLEEP DEPRIVATION AND EXERCISE TOLERANCE

ANNUAL SUMMARY REPORT

BRUCE J. MARTIN, PH.D.

JANUARY 1984

SUPPORTED BY:

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, Maryland 21701-5012

CONTRACT NO. DAMD17-81-C-1023

INDIANA UNIVERSITY SCHOOL OF MEDICINE

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Fifty hours of sleep loss failed to alter thermoregulation in severe cold stress: subjects chose identical work rates to minimize fatigue and cold sensation. Two nights of fragmented sleep worsened mood during exercise but produced no measured change in physiological responses to work or in stress hormone levels. The results suggest that sleep loss is not a true physiological stress but instead manifests itself primarily as a psychological stress.

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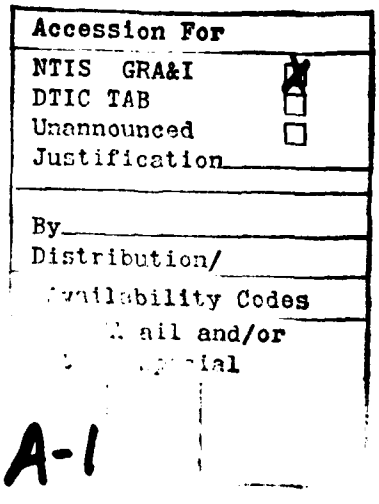

FORWARD

For the protection of human subjects the investigator(s) have adhered to policies of applicable Federal law 45 CFR 46.

BODY OF REPORT

Statement of the Problem:

Sleep deprivation occurs commonly in both civilian and military contexts. Despite this, little is known of the effects of sleep loss on physiological function. Such knowledge could become particularly useful when severe exercise occurs after sleep deprivation.

Background:

Previous work by us and others (1-6) reveals that sleep loss hinders performance of long-term heavy exercise in roughly dose-dependent fashion, without changing any measured aspect of the physiological response to work. These results indicate that sleep loss may act primarily on the mind to produce what are ultimately manifested as psychological changes.

Approach to the Problem:

From these previous findings we moved to investigation of the effect of sleep loss on behavioral (here represented by exercise) responses to the cold. Because the decision to combat cold with heavy work implies a willful decision to work hard, we hypothesized that sleep loss might reduce the work rate chosen and lead to more rapid development of hypothermia.

In another approach to the basic role of sleep as a stress, we studied the influence of two nights of fragmented sleep on subsequent exercise. Here, fragmentation of sleep produced representative conditions most commonly encountered by both civilian and military personnel. By measuring mood, perceived exertion, stress hormonal, and metabolic responses to subsequent exercise, we could define any effects of sleep fragmentation.

Results and Discussion:

We found that 50 hours of sleep loss had no effect on the work rate chosen in severe cold stress. Subjects worked at identical rates in the control and sleep deprived conditions, and ceased work and left that environment after equal durations. Thermal sensitivity to both cold and warm water baths, measured over the hand, was also unchanged by sleep loss. In addition, mean skin and core temperature followed similar patterns in the conditions. The lone exception to stress was the body core temperature (measured rectally) which was significantly lower prior to exercise in the sleep deprived condition. This temperature rose to match that measured under control conditions within 15 min of work, however.

These results are at odds with our previous work showing reduced work performance in sleep deprived states. In this study, a severe cold stress in effect provided the motivating factor for heavy work; the work thus produced before cessation was identical under sleep loss and control conditions.

Two nights of randomly interrupted sleep (8 interruptions per night, with at least 10 min wakefulness enforced at each interruption) worsened mood before and during 30 min of exercise at 70% $\dot{V}O_2max$. These mood changes primarily stemmed from a decrease in vigor and an increase in fatigue. In addition, there was a significant elevation in sleepiness (Stanford Sleepiness Scale) prior to exercise after random sleep interruption; this difference was lost 5 and 25 min after exercise began. These changes in mood could not be correlated with any measured physiological change. Instead, oxygen uptake, minute ventilation, and heart rate were identical under the two conditions. Also, blood levels of several hormones, whose levels are indicative of stress, were unchanged by sleep loss after 30 min of exercise. These included norepinephrine, epinephrine, dopamine, cortisol, and β-endorphin.

Conclusions:

Three years of work lead us to conclude that sleep loss, either total or produced in fragmented fashion, does not impair the fundamental metabolic, ventilatory, cardiovascular, or hormonal response to exercise. Sleep loss does, however, lead to rapidly measurable worsening of mood which may explain reduced tolerance of heavy exercise in the face of unchanged physiology. However, these mood changes are not sufficient for an alteration in exercise-related thermoregulatory behavior in cold stress.

Recommendations:

Sleep loss up to 50 hours duration is only a mild inhibitor of exercise tolerance under conditions in which exercise is forced. Cold stress stands as an example of an extreme forcing, and in that condition exercise tolerance is unimpaired. Though data is lacking, it is reasonable to suggest that the mood changes induced by even very mild sleep loss (through sleep interruptions) might produce very large diminution of exercise performed when the decision to exercise at all remains one of choice.

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Publications:

Kolka, M. A., B. J. Martin, and R. S. Elizondo. Sleep loss and behavioral thermoregulation in cold stress. Eur. J. Appl. Physiol. (Submitted).

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